

# Disappearing His deflection

## *Electrophysiological evidence for conduction defect within the His bundle*

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**SUMMARY** A change in the voltage and character of the His bundle deflection following premature atrial stimuli was observed and analysed in 5 of 95 patients having intracardiac conduction studies because of AV conduction disturbances. Of these 5 patients, 3 had spontaneous block within the His bundle, 2 of them showing block in other segments of the conduction system. With increasing prematurity of programmed atrial stimuli, there was a progressive decrease in the voltage of the His deflection, followed by a split His deflection, and finally disappearance of the His deflection. The voltage of the His deflection was also reduced in sinus beats following spontaneous His bundle premature beats. Similarly, during atrial stimulation at increasing rates, the His deflection decreased in voltage, split, and finally disappeared, but when Wenckebach periods appeared the His deflection reappeared in the first paced beat after the dropped beat. The preceding H-H interval was the only electrophysiological variable consistently related to the changes in the His deflection.

These changes in His deflection can be explained electrophysiologically as the result of a conduction disturbance within the His bundle. The clinical significance of the phenomenon is discussed. The occurrence of this phenomenon during a conduction study makes it difficult or even impossible to localise the AV block precisely.

His bundle recordings have proved useful in the localisation of atrioventricular conduction disturbances. By the use of this method intra-atrial, AV nodal, His bundle, and infra-His blocks can be differentiated. Conduction disturbances within the His bundle are recognised from the widening of the H deflection to more than 25 ms and from the split His phenomenon (Rosen *et al.*, 1971, 1972; Puech and Grolleau, 1972; Bharati *et al.*, 1974; Puech, 1975). Furthermore, block is likely to be located in the common bundle when the HV interval is prolonged with normal QRS configuration, since the coincidence of an identical conduction delay in the right bundle-branch and in the anterior and posterior divisions of the left bundle-branch is highly improbable (Touboul and Ibrahim, 1972).

We present here 5 cases in which a decrease in amplitude, splitting, and disappearance of the His deflection, with increasing prematurity of the atrial extrastimulus, provided evidence for His bundle conduction disturbances.

### Methods

Intracardiac and His bundle electrograms were obtained by the usual method first described by Scherlag *et al.* (1969) and Damato *et al.* (1969). Under fluoroscopy a tri- or quadripolar catheter with interelectrode distances of 10 mm was introduced via the femoral vein across the tricuspid valve to record the His bundle deflection. In addition to electrographic and fluoroscopic control, correct positioning of the catheter was validated by local His bundle stimulation as described by Narula and Samet (1969) and Narula (1975a). The A and V deflections in this tracing were regarded as originating in the low right atrium and right ventricle, respectively. One or two additional tri- and quadripolar electrocatheters were placed for recording and pacing in the high right atrial position (in the vicinity of the sinus node) via the same femoral and/or the antecubital vein. In some cases the His bundle deflection was simultaneously recorded by two different electrocatheters. Simultaneously with 3 surface electrocardiographic leads, filtered bipolar

intracardiac electrograms were recorded with paper speeds of 50, 100, and 250 mm/s (filter specification: high cutoff 400 Hz, low cutoff adjustable between 0.2 and 100 Hz, db: 18). Atrial stimuli were delivered in the high right atrial region using impulses of 0.3 to 0.5 ms in duration and twice diastolic threshold value. Stimulation rate was increased progressively with increments of 10/min. For programmed premature stimulation, single stimuli were given after every eighth spontaneous beat, starting in the atrial refractory period and progressively increasing the A-St interval by 10 ms steps until the basic A-A interval was reached, after which the programmed stimulation was repeated in the opposite direction.

Hellige devices were used for amplifying, filtering, and recording, and a Biomedix diagnostic pulse generator was employed for stimulation.

## Patients

During the 6 months of this study, His bundle recordings were obtained in 95 patients. Sixty-three of these had spontaneous conduction disturbances recognised on surface electrocardiograph leads; in 2 additional patients, first degree block (intra-atrial in one and His bundle block in the other) could be shown by intracardiac recording. Among these were 3 patients with overt signs of His bundle block and 2 patients with left bundle-branch block and prolongation of HV interval, who showed a progressive decrease in amplitude, splitting, and disappearance of the His bundle deflection with increased prematurity of the programmed atrial extrastimulus.

## Results

### CASE 1

This 40-year-old man was referred for His bundle recording because of short runs of tachycardia, occurring mostly during exercise, for the past year. He had had no previous heart disease. His resting heart rate was 64/min and on an exercise test heart rate did not exceed 115/min at 200 watts. After exercise he had frequent ventricular and atrial premature contractions, and then a short run of junctional tachycardia at a rate of 144/min. Intracardiac conduction intervals were A-A 55 ms, A-H 78 ms, H-V 40 ms, H deflection 28 ms. During His bundle recording, junctional tachycardia could be repeatedly triggered and terminated with single atrial stimuli. In this particular patient with evidence of first degree His bundle block (widening of H), the His deflection decreased in amplitude, split, and disappeared with increasing prematurity of the

atrial extrastimulus during programmed stimulation. In addition to the disappearance of the His deflection, aberrant conduction (LBBB pattern) appeared when the A-St interval was 380 ms or less (Fig. 1). Sinus beats following interpolated supra-ventricular premature contractions with retrograde sinoatrial block also showed a decrease in amplitude or disappearance of the His deflection. Before extra-stimuli were sufficiently early to cause AV block, the His deflection had already vanished; while this made it impossible to localise the block accurately, it seems likely that this was within the bundle of His.

### CASE 2

This 51-year-old man was admitted complaining of ill-defined chest pain and with right bundle-branch block of recent onset. The latter was discovered when he was examined on account of weakness after a long ski run 4 months before admission. The resting electrocardiogram showed regular sinus rhythm at a rate of 72/min with a PR interval of 160 ms, complete right bundle-branch block, and a broad Q wave in leads III and aVF indicative of an old inferior scar.

In addition to right bundle-branch block, the His bundle recording showed a prolonged H-V interval indicating a conduction disturbance within the specialised ventricular conduction system.

Atrial pacing with the extrastimulus method showed a progressive H wave voltage decrease which occurred at 600 ms A-St interval and disappearance of the H deflection at an A-St interval of 490 ms. Finally AV block appeared at an A-St interval of 390 ms.

With rapid high right atrial stimulation the H deflection disappeared at a rate of 140/min. Long Wenckebach periods occurred at an atrial rate of 160/min. During Wenckebach periods, the H deflection reappeared regularly in the first beat after the dropped beat, thereby making it possible to localise the Wenckebach block in the AV node (Fig. 2). The H deflection reappeared consistently at an atrial stimulation rate of 180/min with 2:1 AV nodal block. A short run of junctional tachycardia was also recorded on the intracardiac electrogram.

### CASE 3

This 69-year-old woman was studied because of increasing dizziness, a short period of faintness, and atypical chest pain, in May 1976. An electrocardiogram showed sinus rhythm with a rate of 58/min, left bundle-branch block, and a long PR interval (280 ms).

The intracardiac conduction study localised the first degree AV block below the His bundle, with HV interval 130 ms. In addition, there was a short

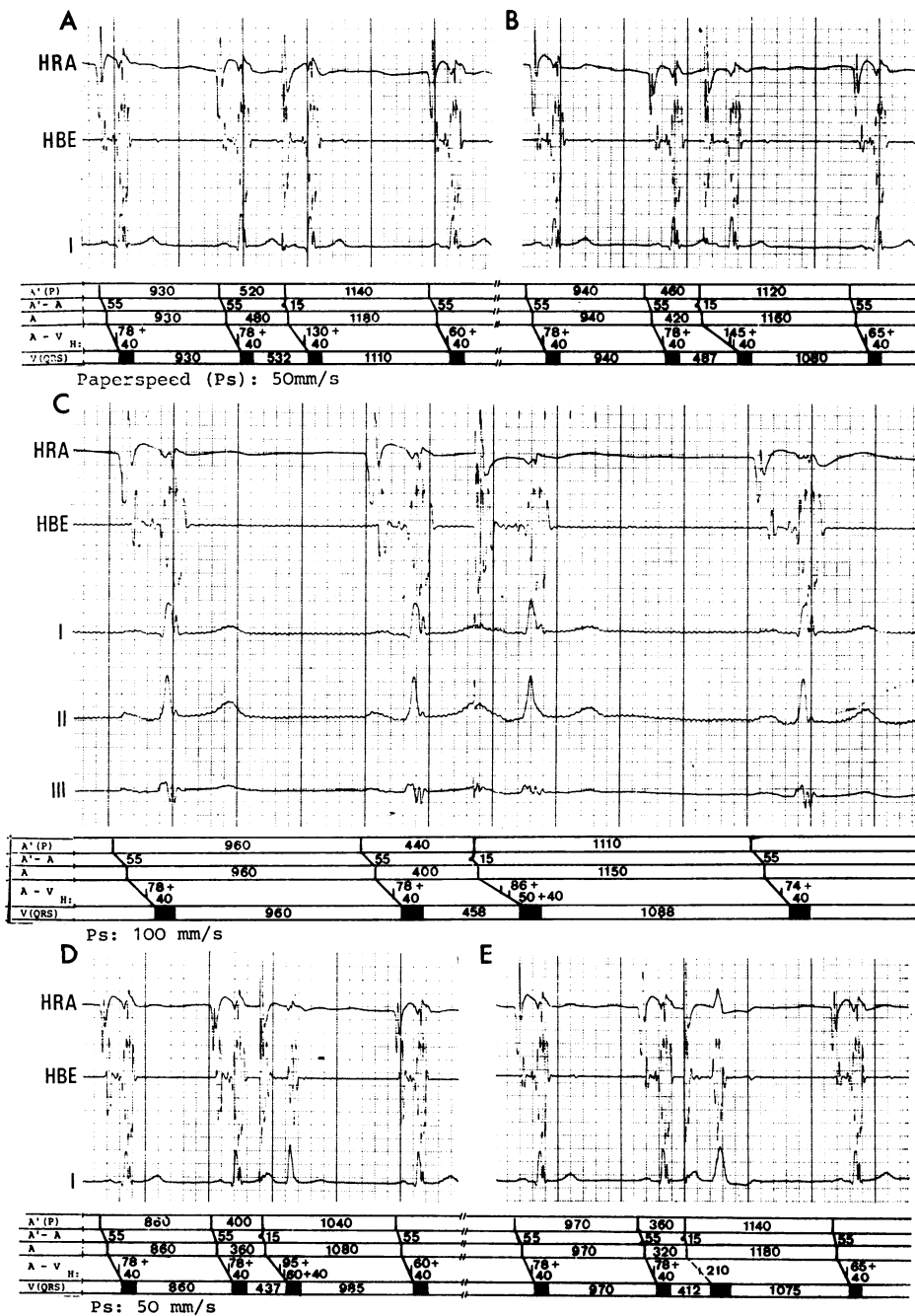


Fig. 1 Case 1: Effect of premature atrial stimulation on voltage and character of His deflection in a patient with spontaneous His bundle block (H wave 28 ms). (A) No change of H deflection at a prematurity of 520 ms; (B) decrease in voltage of H deflection at a prematurity of 460 ms; (C) split His at a prematurity of 440 ms; (D) and (E) hardly recognisable H deflection probably with split at a prematurity of 400 and 360 ms, respectively. In E, LBBB pattern is seen.

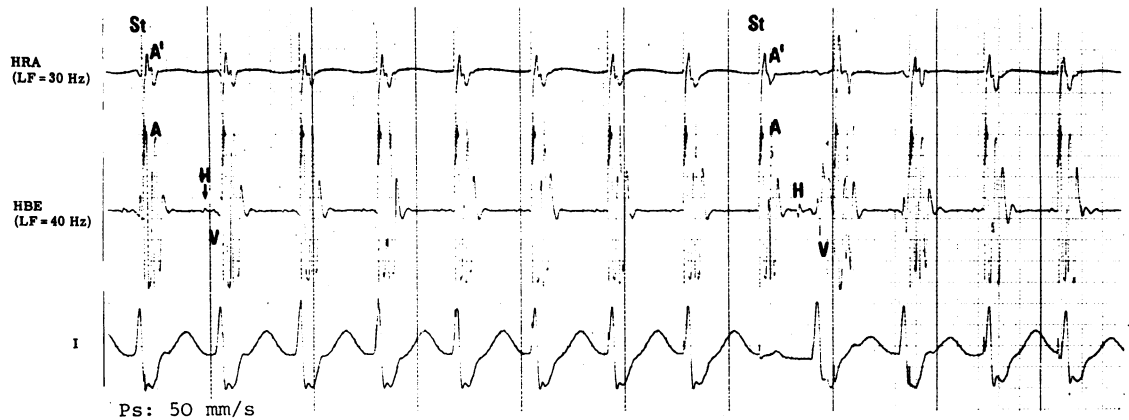


Fig. 2 Case 2: The His deflection disappears during atrial pacing at a rate of 160/min. The atrial stimulation with accelerated rate also provokes long Wenckebach periods in the AV node. Following the dropped beat the His deflection reappears in the first beat of the succeeding Wenckebach period.

run of third degree infra-His block during the His bundle recording. During programmed stimulation there was a progressive decrease in amplitude of H deflection as the A-St interval was reduced from 560 to 430 ms. At 430 ms a split H appeared and was

recognisable until the A-St interval reached 380 ms. At shorter A-St intervals (as in case 1) there were only indirect signs of AV conduction, viz A was followed by V without any recognisable deflection between the atrial and ventricular deflections until

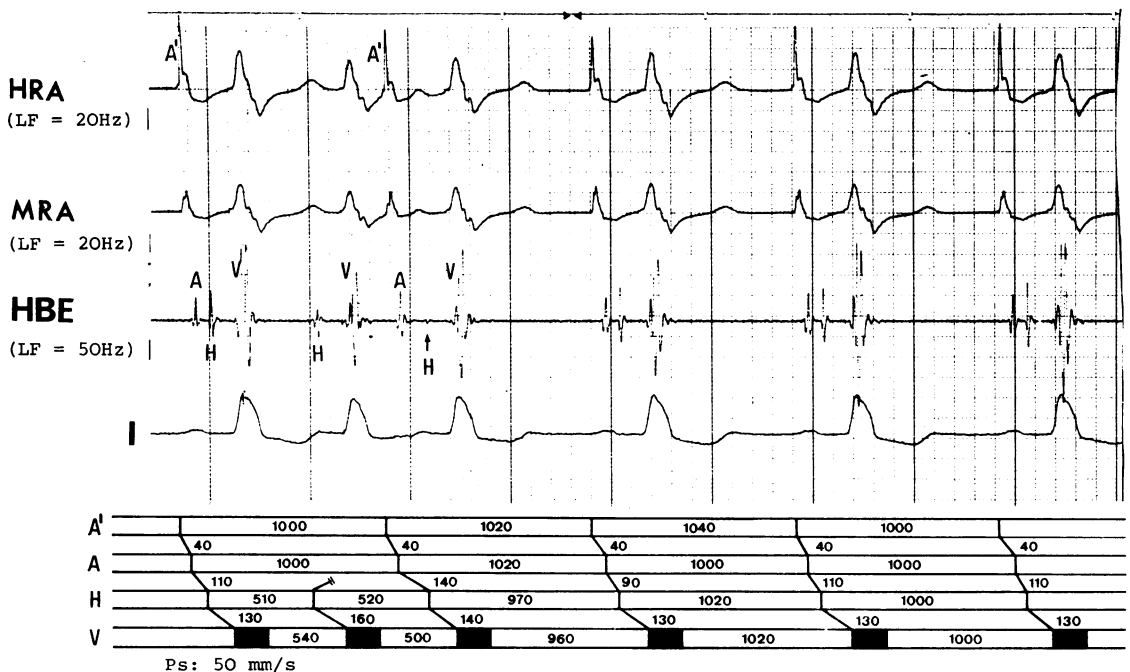


Fig. 3 Case 3: Trifascicular block: left bundle-branch block with HV interval prolongation. The second beat is a H is bundle premature beat which does not disturb the normal sinus rhythm. The His bundle deflection decreases significantly in the succeeding normal sinus beat (3rd beat) as a sign of block within the His bundle.

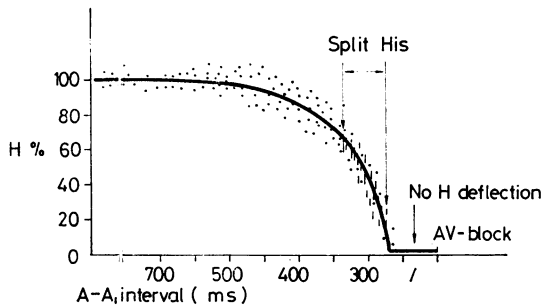


Fig. 4 Case 3: Correlation between the prematurity of atrial stimulation and voltage of His deflection of atrial premature contractions expressed as percentage of the His deflection voltage of the preceding and succeeding beats (128 observations). Vertical lines denote split His deflections.

a block occurred between A and V at A-St interval 320 ms. On asynchronous atrial stimulation at an accelerated rate of 130/min, Wenckebach periods occurred within the AV node. As in case 2, no H deflection was visible from the second beat of the Wenckebach period, but in the first beat following the dropped beat the H deflection reappeared, then disappearing again until the first beat after the next dropped beat. On atrial pacing at a rate of 154/min 2:1 AV block developed. This AV nodal block protected the bundle of His from impulses at the high atrial rate and the H deflection reappeared.

In this particular case we also saw several premature beats originating in the His bundle, which were conducted to the ventricles without change in the QRS configuration, but blocked in retrograde direction by concealed conduction within the AV node. These premature His beats shortened the H-H interval preceding the next normal sinus beats which always showed an H deflection with decreased voltage (Fig. 3). By measuring the voltage of the H deflection in 128 cycles and expressing this as a percentage of the voltage of the H deflections of the preceding and following beats, the curve shown in Fig. 4 was obtained. This case was interpreted as showing first degree block in the bundle of His, a constant first degree and intermittent third degree block in the right bundle-branch in the presence of left bundle-branch block, that is trifascicular block. The patient had a pacemaker implanted and has been free from symptoms since that time.

#### CASE 4

This 66-year-old woman was admitted for a His bundle study because of a history of paroxysmal tachyarrhythmias over an 8 to 9 year period. The onset was insidious and over the preceding 6 months her attacks of tachycardia had become more frequent.

The resting electrocardiogram showed regular sinus rhythm at a rate of 75/min with PQ 195 ms,

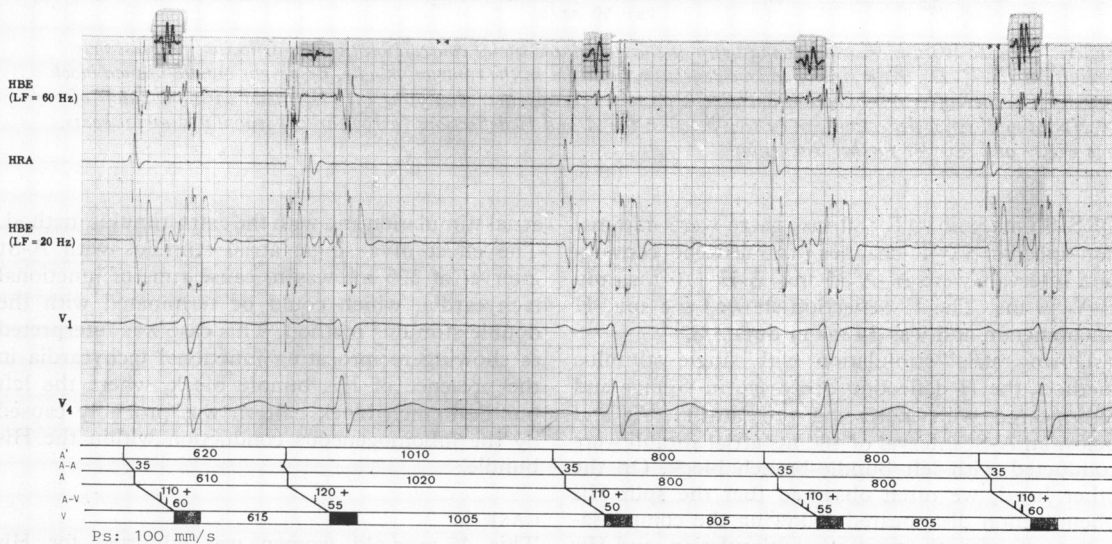


Fig. 5 Case 4: Closely split His deflection during normal sinus rhythm (1st beat). The second beat is a stimulated atrial premature contraction in which the amplitude of the H deflection significantly decreases. Thereafter the compensatory pause gives time for a more effective recovery in the His bundle and its deflection becomes normal in the third beat. In the fourth and fifth beats the split His deflection progressively redevelops.

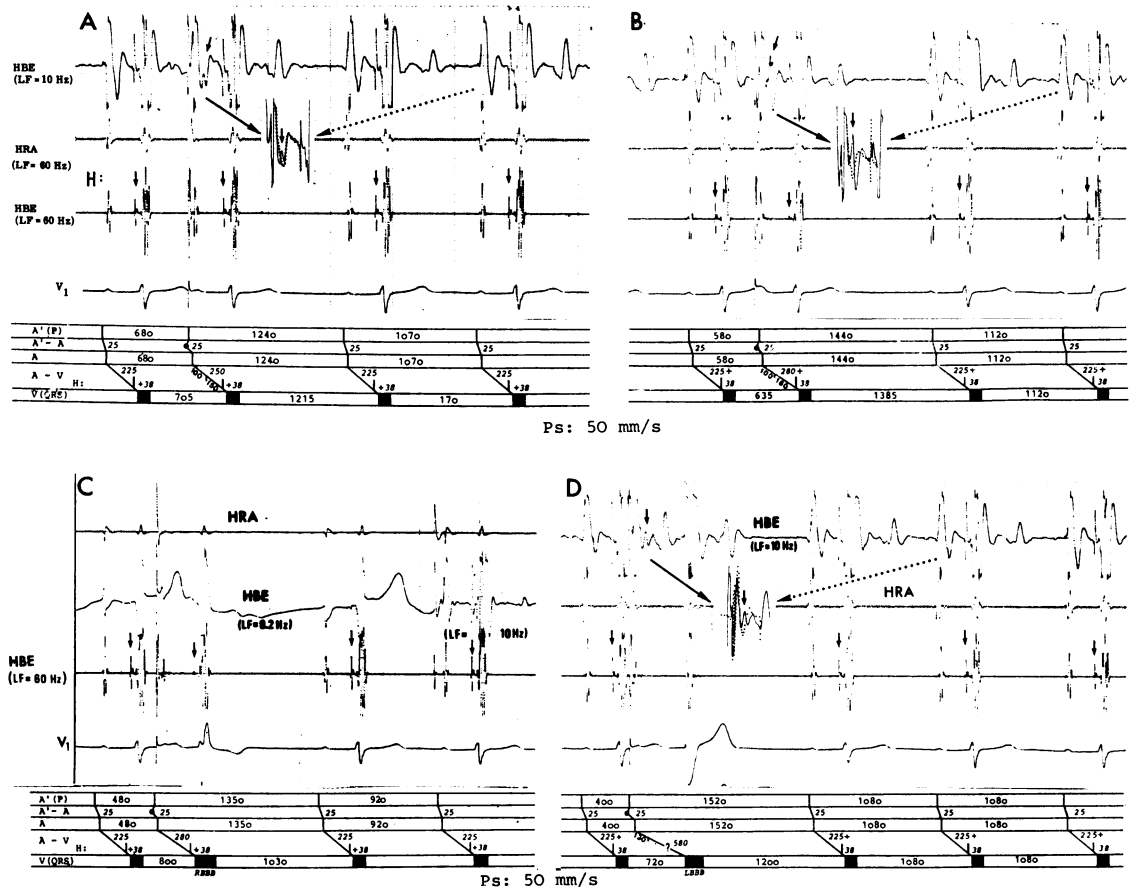


Fig. 6 Case 5: With increasing prematurity of the stimulated atrial contraction the His deflection progressively diminished (B, C, D). As a sign of inhomogeneous conduction in the common bundle, first right bundle-branch block pattern (C), thereafter left bundle-branch block pattern (D) appears. Analysing the HBE lead with low cutoff of 10 Hz an additional deflection can be seen between the A and H deflections (arrow) in the stimulated atrial beats. See Fig. 7 and text for further discussion.

QRS 80 ms, and QT 380 ms; there were minimal non-specific ST-T changes. Intracardiac conduction intervals were A'-A 35 ms, A-H 110 ms, and H-V 50 ms. The H deflection showed the split H phenomenon and was 34 ms in duration.

During atrial stimulation with single stimulus method, the H deflection decreased in voltage and disappeared with increasing prematurity. At and below an A-St interval of 480 ms atrial stimuli were conducted with left bundle-branch block. On the other hand, we often observed that the split His phenomenon disappeared following the compensatory pause after the premature stimulation, and His deflection becoming normal in form and duration for that single beat (Fig. 5). Again the voltage of the His deflection showed a direct relation to the length of the preceding H-H interval both during rapid

atrial stimulation and with the extrastimulus method. The effect of a single atrial stimulus with A-St interval of 325 ms was to cause runs of junctional tachycardia, which could be terminated with the double stimulus method. This case was interpreted as showing reciprocating junctional tachycardia in the presence of His bundle block, where the left bundle-branch block pattern was probably caused by the inhomogeneous conduction within the His bundle.

#### CASE 5

This 46-year-old woman was admitted for His bundle electrogram because of a 4-year history of bouts of tachycardia, vertigo, and syncope on getting up in the morning. Her symptoms had increased slowly in intensity over the years. The

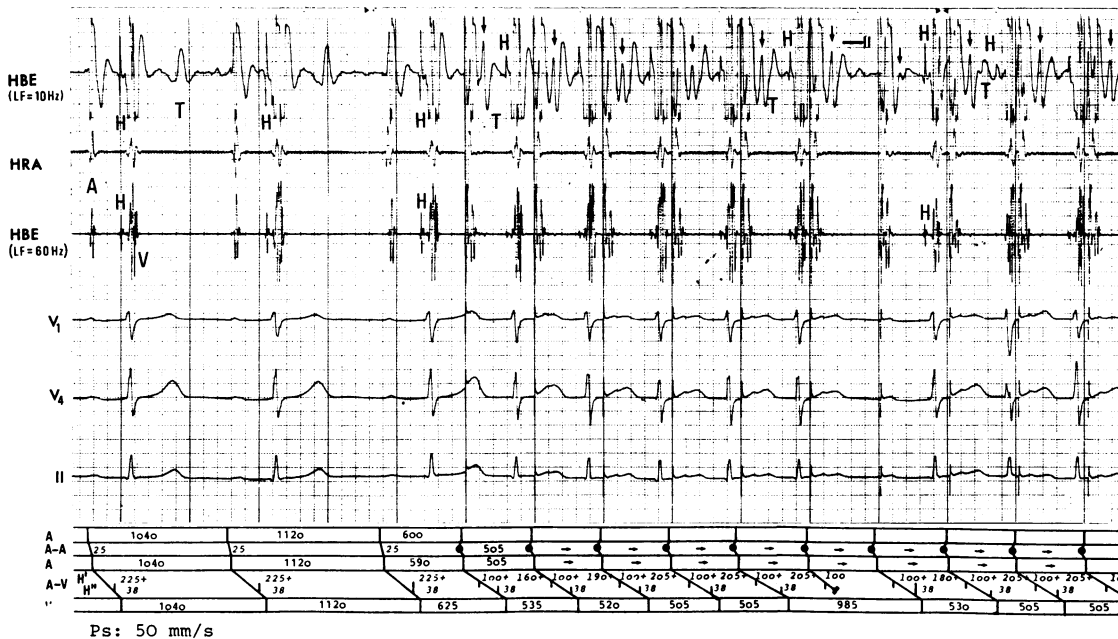


Fig. 7 Case 5: A long period of second degree AV block develops during atrial stimulation at a rate of 118/min. The H deflection decreases during atrial stimulation and increases after the dropped beat in the conventional HBE lead. In another HBE lead with low electric cutoff an additional deflection ( $\downarrow$ ) appears 100 ms after the stimulated A. The progressive prolongation of PR and  $\downarrow$ -H intervals in the first two paced beats (4th and 5th beats) can be explained by the prematurity of the stimuli. Conduction in the next 3 beats is unchanged, suggesting that the block appearing after the additional deflection seen only in stimulated beats should be regarded as a Mobitz type II block within the His bundle. This deflection ( $\downarrow$ ) should not be regarded as the first component of split His deflection as its unchanging distance from the preceding A wave makes this interpretation improbable.

resting electrocardiogram showed sinus rhythm at a rate of 56/min with first degree AV block (PR 290 ms, QRS 80 ms, QT 420 ms). The QRS axis was  $+30^\circ$  and QRS configuration within normal limits. Intracardiac conduction intervals were A'-A 25 ms, A-H 225 ms, and H-V 38 ms. The duration of the H deflection was 28 ms.

On the resting electrocardiogram there were episodes of SA block with junctional escape beats giving rise to an irregular rhythm. Sinus node recovery time was measured 8 times during the His bundle recording, the maximal prolongation being 4220 ms after rapid atrial stimulation at 150/min for 120 s. On 4 occasions sinus arrest followed rapid atrial stimulation and a junctional escape rhythm took over with an escape interval of 1800 ms and a rate of 44/min. On another occasion, sinus arrest was associated with a ventricular escape rhythm at a rate of 32/min which appeared 2400 ms after cessation of atrial stimulation. With programmed atrial stimulation the His deflection was the same as in sinus rhythm at an A-St interval 660 ms. However, with A-St interval 560 ms the H deflec-

tion became smaller and wider but conduction into the ventricle remained normal. At 450 ms the H deflection was barely discernible and conduction took place only through the left bundle-branch (right bundle-branch block pattern). At A-St intervals of 450 and 400 ms the H deflection decreased further and disappeared, respectively, and conduction then was only through the right bundle (left bundle-branch block pattern) (Fig. 6). Atrial pacing at a rate of 120/min provoked periods of second degree AV block. Here again the His deflection became smaller and then increased to its former amplitude following the dropped beats of the Wenckebach periods in the His bundle recording with filter set for 60 Hz low cutoff. In this particular case, the His recording with filter set for 10 Hz low cutoff showed an additional deflection at high rates and during premature atrial stimulation. Its distance from the preceding A wave was shorter than the A-H interval in sinus rhythm and did not change with either prematurity or rate of stimulation (Figs 6 and 7). Block causing the dropped beat of the Wenckebach periods occurred between this low rising deflection and the His deflection (Fig. 7).

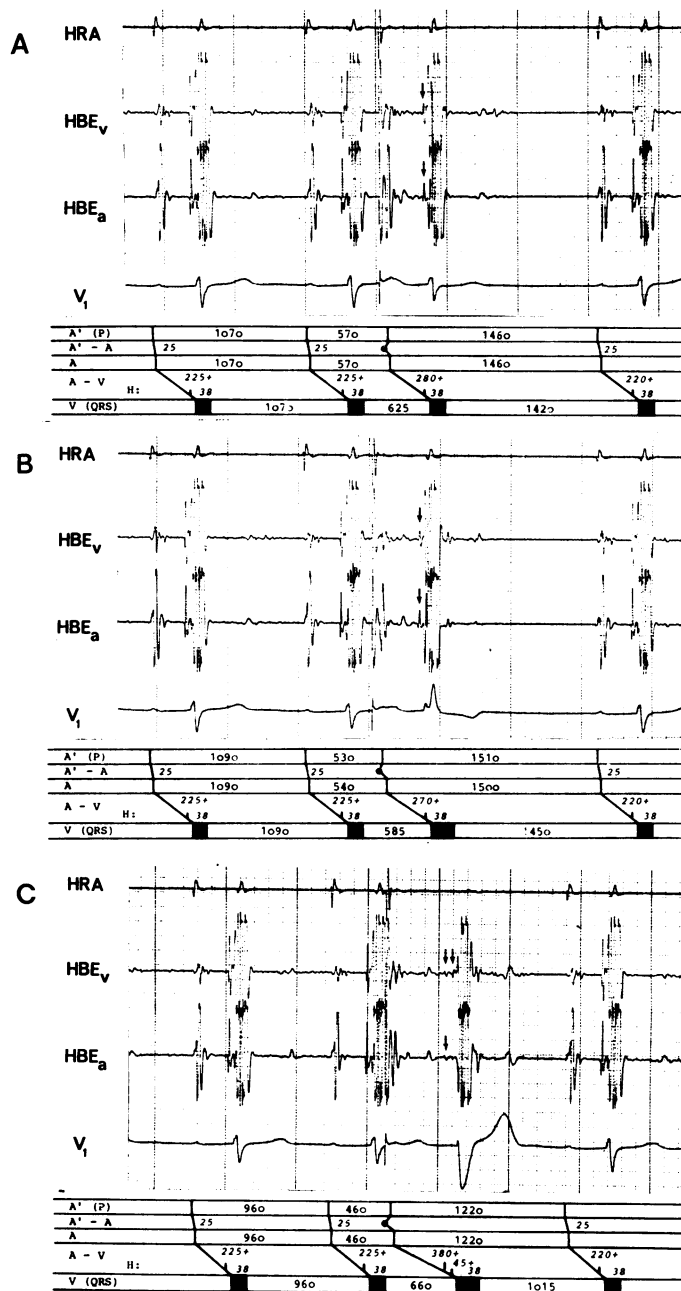


Fig. 8 Case 5: With premature stimulation, the His deflection changes in identical fashion in each of the two simultaneous bipolar His bundle electrograms (HBE) which were recorded by the two distal (ventricular HBE, HBE<sub>v</sub>) and two proximal (atrial HBE, HBE<sub>a</sub>) poles of a quadripolar catheter. Note the difference in voltage of A in the two tracings. With premature stimulation at 460 ms a split His occurs (C); the distal H deflection is clearly recognisable only in HBE<sub>v</sub>. Arrows show His deflection in stimulated beats.

## Discussion

Accepted as criteria for block within the His bundle are widening of the His deflection, its duplication (split H), and first, second, or third degree blocks below the H deflection with narrow QRS configuration (Rosen *et al.*, 1971, 1972; Puech and Grolleau,

1972; Touboul and Ibrahim, 1972; Bharati *et al.*, 1974). Block in the bundle of His is commonly associated with conduction disturbances at other levels in the conduction system. Puech (1975) reported on 383 cases of spontaneous AV block. His bundle involvement was present in 68 cases. In 22 of these 68 patients (32%) His bundle block was



associated with conduction disturbances below the His bundle (wide QRS). On the other hand 13 per cent of his 173 cases of intraventricular conduction disturbance showed additional His bundle block. In 72 per cent of Narula's (1975b) 122 cases of first degree AV block delayed conduction occurred in multiple segments of the AV conduction system. These studies indicate that in case of AV block, involvement of more than one segment of the conducting system is common.

In 4 of our 5 cases spontaneous conduction disturbances were present, and in 3 cases the His bundle electrogram showed first degree block within the common bundle.

During premature stimulation a decrease of voltage, a duplication, and finally disappearance of the His deflection were observed, this phenomenon being repeatedly reproducible in these patients. Because of the method of His bundle electrography, any change in the amplitude and shape of the His bundle deflection has to be carefully interpreted. In the opinion of Narula (1975a) these changes should not be taken to indicate an alteration of conduction in the His bundle. He attributed reduction in amplitude of the H deflection with premature atrial stimulation to dislocation of the catheter by anomalous atrial contraction. But if the premature stimulation shown in his figure was able to dislocate the recording electrode, we must conclude that the following sinus beat restored it to its previous position. We could accept this explanation for a decrease or disappearance of the His potential in single instances, but generally our experience does not confirm this view as we have always seen fluctuating changes in the His deflection when caused by electrode movement. In our reported cases the change of voltage of the His deflection always took place suddenly and was a function of the preceding H-H interval, which was unrelated to the site of origin of the beats showing this change. The amplitude of the His bundle deflection decreased progressively with the shortening of the A-St interval during programmed stimulation. Furthermore disappearance of the H deflection was preceded in 3 of these 5 patients by its splitting (Figs. 1 and 8). A change in the site of origin of the atrial contraction had no effect on the His deflection; thus, the same His deflection was present during atrial stimulation and normal sinus rhythm. When premature stimuli were delivered during the paced rhythm, the decrease in size of the H deflection was the same as in the premature beats in sinus rhythm. A diminution of the H deflection was also seen in sinus beats if they came early following stimulated or spontaneous atrial or His bundle deflections, as in Fig. 3. When two separate His bundle recordings

were obtained using adjacent poles of a multipolar catheter, changes in the voltage of the His deflection were identical in the two tracings (Fig. 8). The presence of frequent His bundle premature beats and junctional tachycardias in our cases are additional evidence for a lesion in the common bundle, as has been emphasised by others (Puech and Grolleau, 1972; Narula, 1973). At a stimulation rate provoking Wenckebach periods in the AV node, rate, origin, and pattern of atrial contraction remain constant, but as shown in Fig. 2 this rate caused the disappearance of the H deflection, which reappeared again in the first beat following the dropped His-Purkinje deflection. At a higher atrial stimulation rate with 2:1 AV block, His bundle deflections reappeared at a slower rate below the block, proving that its presence does not depend on rate and pattern of atrial contraction but on the time available for repolarisation of the common bundle. Illustrations showing the same phenomenon of decrease in amplitude of the His deflection during premature atrial stimulation can be found in previous publications but, as far as we know, have not been interpreted as showing a conduction defect within the His bundle. Some examples are Figs. 4—2, 4—3, 6—6, and 6—16 in the recent book by Lister *et al.* (1976), three figures in Narula's book (1975c) (Chapter 10 Fig. 9, Chapter 14 Fig. 11b, and Chapter 14 Fig. 2). In the excellent Fig. 31 of Damato *et al.* (1975), the first premature stimulation was followed by a decrease in amplitude of the His deflection, by an extreme prolongation of H-V interval, and by a left bundle-branch block pattern. Following the second stimulation in the same figure, the His deflection is even smaller, the H-V prolongation is less, and the QRS complex shows a right bundle-branch block pattern, as in our case 5. In contrast to these findings, Fig. 4.32 of Puech (1975) showed, in a patient with left bundle-branch block, a sudden increase in voltage of His potential, shortening of the H-V interval, and disappearance of the left bundle-branch block for a single beat during a Wenckebach period. Puech noted this change in the His deflection in the legend to his figure, but did not explain it.

It is necessary to find an electrophysiological explanation for the inability to record a His deflection in atrial premature beats in these patients, especially as 4 of our 5 patients had manifest signs of conduction disturbance within the His bundle (widening of the H deflection, H-V prolongation with normal QRS, split H, and His bundle premature beats), accompanied in 3 cases by block in other segments of the conducting system.

An example of conduction without a detectable electrographic sign is the spread of an impulse in

the AV node. The possibility that the N deflection arises in the AV node has been widely discussed (Hoffman *et al.*, 1960; Damato and Lau, 1969; Damato *et al.*, 1970; Massumi, 1970; Brodsky *et al.*, 1971; Mendez and Moe, 1972; Kupersmith *et al.*, 1973; Narula, 1973), but in agreement with most authors (Hoffman *et al.*, 1960; Mendez and Moe, 1972; Kupersmith *et al.*, 1973; Narula, 1973; Narula, 1975a) we believe that it originates not in the AV node but in intra-atrial tracts and label it Tr, as proposed by Narula. Some authors attribute the absence of an AV node deflection to potentials too small to be recorded. According to Hoffman *et al.* (1960), in tissues such as the AV node with slow rise of depolarisation ( $dv/dt$ ), the extracellular potential difference between the electrodes will be too small to give rise to an electrographically recordable signal.

Hecht (1957), Vaughan Williams (1959), and Spach *et al.* (1972) have noted that extracellular potential of cardiac fibre resembles the second derivative of the rate of rise (phase 0) of action potential. This finding means that with decreasing the rise of phase 0 depolarisation highly decreases the extracellular electrographic sign of this process and also explains the lack of direct electrographic evidence of the impulse conduction in sinus and AV node.

Paes de Carvalho (1962) writes: 'Activity which spreads at 0.05 m/s and takes 30 ms to attain maximum depolarization at given spot is not likely to show up in extracellular recordings especially if the size of the electrodes or the frequency response of the apparatus are not in keeping with the slowness of the process.' This statement was confirmed by Kupersmith *et al.* (1973) who placed electrodes directly on the AV node in 58 patients during open heart surgery, but could not demonstrate any recognisable activity of the node. However, in their study only electric signals below 12 Hz were filtered out in contrast to the usual intracardiac electrographic technique using a low filter of 40 to 60 Hz. As it was possible that slowly rising His bundle depolarisations had been filtered out in our patients, we repeated the programmed stimulation in 4 cases (cases 2 to 5) filtering out simultaneously on one channel the signals below 60 Hz, on another decreasing progressively the filter level to 0.2 Hz. In 3 of the 4 cases we obtained no additional information by this technique, but in the last case an additional deflection appeared in the His bundle tracing in the stimulated atrial beats with low (10 Hz) filter cutoff (Figs. 6 and 7). Since this followed A by a fixed coupling interval it could be related to atrial depolarisation, though its exact origin was uncertain. A split His phenomenon cannot

be excluded, and it also may reflect afterswing of the recorder unit due to electric resonance (see also legend to Fig. 7). The AV block during atrial stimulation at high rates was located between this slow rising potential and the His deflection (Fig. 7). During premature stimulation, the His potential disappeared in this lead before the AV block appeared (Fig. 6).

We suggest that in these cases conduction within the His bundle closely resembled that in the AV node, this electrographic similarity being the result of an electrophysiologic similarity. It is well known that premature impulses (spontaneous or paced) can meet a stage of incomplete repolarisation. The onset of an action potential at a lower level of membrane potential results in a slower phase 0 depolarisation and slower conduction velocity. These changes of membrane events will appear as prolongation of the conduction interval, concealed, and decremental conductions. This also means that in an extracellular recording there is only a quantitative difference between widening and disappearance of an electrographic deflection, in this case a His deflection. According to James (1976), the His deflection arises from the P cells of the bundle. In animal experiments, he produced an intermittently split H deflection, explaining the splitting by dissociation of P cells, and the reconnection of the two components of H by their 'regluing'. These experiments do not explain why tissues containing even more P cells do not show electrographic signs of depolarisation, but show that in cases with split H there is a silent zone between the upper and lower part of the common bundle.

Normally we cannot produce any recognisable change in His bundle conduction by atrial stimulation since the conduction quality of the common bundle is superior to that of the AV node, so that the latter functions as the 'physiological gate' to the whole AV conduction process. But, when conduction in the His bundle decreases to or below the level of that of the AV node by virtue of a change in its membrane potential and form of action potential, it will take over the role of the gate. Thus, a first degree block within the His bundle can be recognised either by the well-known signs of His bundle block or only by testing the bundle with premature and high frequency stimulation. Our clinical findings are supported by Myerburg *et al.* (1970) who described the progressive change of action potential along the His bundle and Purkinje system. Benitez *et al.* (1973) showed that this progressive increase in rate of rise of action potential along the His bundle can be abolished by inhibiting the fast inward current by a KCl solution of 29.3 mmol/l or by tetrodotoxin so that its action potential becomes

similar to that of AV node. Their model experiments provide an electrophysiological explanation of our clinical observations which on the other hand support the sinoventricular conduction concept of Sherf and James (1969).

The clinical significance of our present findings is related to the localisation of AV blocks. Thus when an A deflection is not followed by H and V the block cannot always be localised in the AV node. The site of block is clear if both H and V, or V only, disappear suddenly with increased prematurity of the atrial stimulus. But if the disappearance of the H deflection is preceded by its progressive decrease in voltage, AV conduction is proven only indirectly by the fact that A is consistently followed by V. In such cases the supra-, intra-, or infra-Hisian localisation of the site of the AV block is impossible. Additional tests (rapid atrial pacing, direct His bundle pacing, recording the His bundle electrogram with different filtrations for detecting slow rising potentials, examination of retrograde conduction, etc.) have to be performed to determine the exact localisation of the AV conduction defect.

An unsuccessful attempt to record a His deflection is not necessarily a technical failure. It can also be a sign of His bundle block as was seen in an additional case of ours with intermittent bifascicular block and Adams-Stokes syncope. During the electrophysiological study we had difficulty in recording an H deflection, but this finally appeared in the form of a split H or a single H deflection of 40 ms duration for a few beats only and then disappeared again. Nevertheless we suggest that a diagnosis of His bundle block should be made on the basis of the absence of an H deflection only if its absence is proved conclusively by approaching the His bundle from different directions (superior or inferior vena cava, coronary sinus, aorta).

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